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EFFECT OF LEPIDIUM SATIVUM DIETARY SUPPLEMENT ON BIOCHEMICAL ALTERATIONS IN CHICKEN CAUSED BY SYNTHETIC PYRETHROID TYPE II FENVALERATE

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ABSTRACT

Synthetic pyrethroids, particularly Type II fenvalerate, are widely used in pest control but pose significant toxicological risks to poultry. This study investigates the protective effects of *Lepidium Sativum* (garden cress) as a dietary supplement in mitigating hemato-biochemical alterations induced by fenvalerate exposure in chickens (Gallus domesticus). A controlled experimental design was conducted, wherein chickens were divided into four groups: control, fenvalerate-exposed, *Lepidium Sativum*-supplemented, and a combination of fenvalerate exposure with *Lepidium Sativum* supplementation. Biochemical parameters, serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), and Alkaline phosphatase, Acetylcholinesterase, Total protein, Cholesterol, and Blood glucose were evaluated.

Results indicated that fenvalerate exposure significantly increased hepatic markers and induced adverse biochemical changes, including hepatic dysfunction, disturbances in lipid metabolism, and alteration in acetylcholinesterase activity. However, dietary supplementation with *Lepidium Sativum* markedly alleviated these toxic effects by restoring antioxidant enzyme activities, improving hematological indices, and stabilizing liver function biomarkers.

The findings suggest that *Lepidium Sativum* exerts a protective role against fenvalerate-induced toxicity by enhancing antioxidant defence mechanisms and mitigating biochemical disruptions. This study highlights the potential of natural dietary supplements in safeguarding poultry health against synthetic pyrethroid toxicity.

KEYWORDS: *Lepidium Sativum*, Fenvalerate, Biochemistry, Poultry, Antioxidants

INTRODUCTION

Synthetic pyrethroids have emerged as a pivotal component of modern pest control strategies, renowned for their high efficacy against a wide range of insect pests and relatively low environmental persistence compared to older organochlorine compounds (Thatheyus & Selvam, 2013). Organochlorines, organophosphates, organofluorines, carbamates, pyrethroids, bipyridyl herbicides, triazine herbicides, triazoles, and chloroacetanilide herbicides are among the chemical families of pesticides (Georgiadis et al 2018). However, their widespread use in agriculture and veterinary medicine raises significant concerns about unintended ecotoxicological impacts, particularly on non-target organisms such as poultry. Type II pyrethroids, including fenvalerate, exhibit enhanced stability and potency due to structural modifications like α-cyano groups, which also increase mammalian toxicity by disrupting neuronal sodium channel kinetics (MSD Veterinary Manual, 2022). Poultry exposure to these compounds can occur through contaminated feed and environmental residues, leading to documented hematological disturbances, including anemia, leukocytosis, and alterations in hepatic enzymes indicative of metabolic stress (National Center for Biotechnology Information, 1988).

The toxicity and chronic exposure of synthetic pyrethroid type II fenvalerate in chicks emphasize the necessity of safeguards against fenvalerate-induced toxicity in chickens (Verma 2015). The study of defensive countermeasures has accelerated due to the increasing awareness of synthetic pyrethroid bioaccumulation in food chains. *Lepidium Sativum* (garden cress) exhibits remarkable antioxidant potential, suggesting that natural phytochemicals hold great promise in reducing negative effects due to their distinct glucosinolate and flavonoid composition (El-Saber Batiha et al., 2021).

Lepidium Sativum (LS) is a very useful medicinal seed also known as garden cress (GC). LS seeds an ancient remedial herb that comes under the family Brassicaease. This plant is produced and also found as a natural herb in many areas of India. These seeds are very common in conventional ayurvedic medicine as well as home remedies in various parts of the country for a long time in India (Mali *et al.* 2007). LS seeds are brownish-red in colour, oval in shape, pointed at one end and conical at one end, and with a smooth surface. LS is cultivated as a common plant species in many regions of India. Seeds possess high nutritional advantages. Linolenic acid contributes 18-24% approximately 34% of total fat (Diwakar *et al.* 2010). Oleic and linolenic acid are the primary fatty acids present in LS seed oil, approximately 29.3 wt% to 30.6wt%, respectively, as well as a high concentration of vitamin E. Sitosterol, campesterol and avenasterol are basic phytosterols present in LS seed (Bryan *et al.* 2009). The postnatal period is very crucial for every woman.

LS seeds are incorporated in the diet of lactating women, which in turn stimulates milk secretion. It also contains a high percentage of calcium and Iron also, administration of these seeds is beneficial in curing certain diseases.

The seeds are rich in lysine (7 - 8 g/100g of protein) but have a poor quantity of methionine (0.99 - 1.85 g/100g) and cysteine (0.78 - 1.23 g/100g). A small amount of tryptophan (approx. 1.2 g/100g), phenylalanine (3.89 g/100g) and other amino acids are also found in seeds. They include antioxidants and have antibacterial properties (Cross et al. 2007), and they aid in immune development and growth (Ko et al. 2008). Despite being rich in nutrients, the seeds are not popular among the population. As the seeds are cost-effective, they may be used in ample amounts to provide nutrition to the population. Keeping in view the value of these seeds present study was designed to examine its effect on the biochemical profile of the chicks.

Current mitigation strategies remain inadequate in addressing the subacute toxicity manifestations that compromise poultry productivity and food safety, creating an urgent need for evidence-based nutritional interventions. This investigation evaluates *Lepidium Sativum*'s protective efficacy against fenvalerate-induced hematobiochemical disruptions in Gallus domesticus, examining its potential to enhance oxidative homeostasis through dietary supplementation. By establishing critical dose-response relationships and advancing practical strategies for safeguarding avian health in pesticide-intensive agricultural systems, this study contributes to the development of sustainable and environmentally friendly approaches to mitigate the toxicological impacts of synthetic pyrethroids.

METHODOLOGY

The present study was conducted using day-old broiler chicks procured from local hatcheries. The chicks were randomly and equally divided into seven experimental groups to assess the toxic effects of synthetic pyrethroid Type II Fenvalerate and the potential protective role of *Lepidium Sativum* (LS).

Experimental Design: The seven groups included a control group, a vehicle group, two groups exposed to different concentrations of Fenvalerate, a group receiving only *Lepidium Sativum* seeds, and two groups exposed to Fenvalerate in combination with *Lepidium Sativum* seeds. The treatments were administered for 10 days as follows:

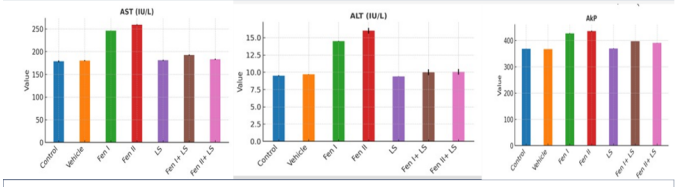
1. Control Group: Received a standard diet supplemented with groundnut oil.
2. Vehicle Group: Received only groundnut oil for vehicle control.
3. Fen I Group: Administered 1% LD50 of Fenvalerate.
4. Fen II Group: Administered 5% LD50 of Fenvalerate.
5. LS I Group: Fed with *Lepidium Sativum* seeds as a dietary supplement.
6. Fen + LS I Group: Administered 1% LD50 of Fenvalerate along with *Lepidium Sativum* seeds.
7. Fen + LS II Group: Administered 5% LD50 of Fenvalerate along with *Lepidium Sativum* seeds.

Preparation and Administration of Feed: For each experimental group, a separate diet was prepared by thoroughly mixing the corresponding quantity of Fenvalerate and/or *Lepidium Sativum* seeds in the feed. Control birds were provided with a standard diet without any additives.

Sample Collection and Analysis: Blood samples were collected from the birds at regular intervals for biochemical analysis. The serum was separated by centrifugation at 3000 rpm for 15 minutes and stored for further biochemical analysis.

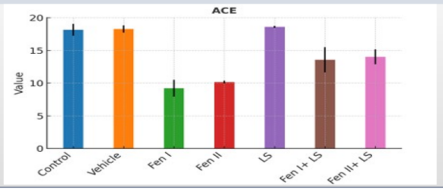
Statistical Analysis: The obtained data were analyzed to evaluate the significance of differences among the groups. Appropriate statistical methods were applied to assess the impact of Fenvalerate toxicity and the protective efficacy of *Lepidium Sativum* seeds.

Effect of Different Treatments on Hepatic Blood Parameters Levels



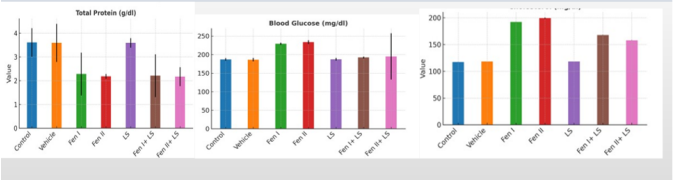
The study demonstrates dose-dependent hepatotoxicity of fenvalerate , evidenced by elevated AST , ALT , and ALP in Fen I/II groups, reflecting hepatocellular injury and cholestasis from oxidative membrane damage.*Lepidium sativum* (LS) attenuated these effects, notably normalizing AST and ALT in Fen+LS groups, with greater efficacy at lower fenvalerate doses Fen I+LS AST/ALT reductions of 22% and 31% vs. Fen I alone). Residual ALT elevation in Fen II+LS suggests LS's antioxidant phytochemicals (e.g., isoflavonoids) partially counteract fenvalerate-induced toxicity.

Effect of different treatments on acetylcholinesterase (ACE) (nmol Ach/mg protein/min) levels



Suppressing acetylcholinesterase (ACE) activity in fenvalerate-exposed chicks and its partial restoration with *Lepidium sativum* (LS) co-administration reflect a dual mechanism of neurotoxic disruption and phytochemical-mediated protection.

Effect of different treatments of Fenvalerate/LS Seeds on Total Protein (g/dl), Blood Glucose (mg/dl) and Cholesterol(g/dl)levels



Cholesterol levels increased significantly in the Fen I (192.21 ±0.10 mg/dl) and Fen II (199.39±0.91 mg/dl) groups compared to the control (117.25±0.05 mg/dl) and vehicle (118.23 ±0.03 mg/dl), indicating dyslipidemia due to fen exposure. LS alone (118.12±0.03 mg/dl) maintained normal cholesterol levels, while co -administration with fen (Fen I + LS: 167.63 ±0.21 mg/dl, Fen II + LS: 157.92±0.05 mg/dl) showed partial but not complete restoration, suggesting a moderate protective effect of LS against fen -induced hypercholesterolemia.

RESULTS AND DISCUSSION

The present study provides significant insights into the hepatotoxic and metabolic effects of fenvalerate, a Type II synthetic pyrethroid, in poultry. The observed elevations in AST,

ALT, ALP, and cholesterol levels in the Fen I and Fen II groups strongly indicate hepatic dysfunction and oxidative stress, which are characteristic of pyrethroid-induced toxicity. These findings are consistent with previous studies demonstrating the hepatotoxic potential of fenvalerate, primarily attributed to reactive oxygen species (ROS) generation, lipid peroxidation, and mitochondrial dysfunction (Raina et al., 2008; Sharma et al., 2019). The hepatocellular injury is likely exacerbated by oxidative stress-mediated apoptosis, which disrupts normal hepatic function.

Groups → Parameters ↓	Control Gr	Vehicle Gr	Fen I Gr	Fen II Gr	LS Gr	Fen I+ LS Gr	Fen II+ LS Gr
Aspartate aminotransferase (AST) (IU/L)	178.53±2.31	180.1±1.21	246.14±0.22[a]	259.19±1.16[a]	181.1±1.21	192.43±1.43	183.18±1.23
Alanine aminotransferase (ALT) (IU/L)	9.5±0.07	9.7±0.02	14.5±0.03[a]	16.01±0.41[b]	9.4±0.02	10.01±0.41	10.06±0.41[b]
Alkaline phosphatase (AkP) (IU/L)	368.43±0.38	367.21±0.20	427.18±1.32	436.08±2.30[a]	369.39±0.66	397.40±0.15[b]	391.33±0.30
Acetylcholinesterase (ACE) (nmol Ach/mg protein/min)	18.16±0.91	18.28±0.56	9.22±1.29	10.17±0.22	18.61±0.15	13.58±1.92	14.03±1.15
Total protein (g/dl)	3.61±0.6	3.59±0.8	2.28±0.9[a]	2.18±0.1[a]	3.59±0.2	2.21±0.9[a]	2.17±0.4[a]
Cholesterol (mg/dl)	117.25±0.05	118.23±0.03	192.21±0.10[a]	199.39±0.91[a]	118.12±0.03	167.63±0.21[b]	157.92±0.05
Blood glucose (mg/dl)	186.81±3.52	186.17±4.51	229.08±3.01	233.62±5.16	187.23±3.44	192.11±2.52	195.05±62.22

Table 1.1 Biochemical alterations due to fenvalerate/*Lepidium Sativum* exposure to the poultry for 10 days

The co-administration of *Lepidium Sativum* led to a partial restoration of AST and ALT levels, suggesting its hepatoprotective role in mitigating oxidative damage. This protective effect is likely due to LS-mediated enhancement of antioxidant defence mechanisms, possibly involving increased activity of endogenous antioxidants. The ability of LS to modulate inflammatory pathways and prevent excessive lipid peroxidation may contribute to its hepatoprotective efficacy, as reported in studies involving other hepatotoxic agents (Gupta et al., 2020).

The study also highlights fenvalerate-induced dysregulation of lipid metabolism, as evidenced by a significant increase in cholesterol levels. Previous research has linked pyrethroid exposure to alterations in hepatic lipid metabolism, potentially due to oxidative stress-driven disruption of cholesterol biosynthesis pathways or inhibition of lipoprotein clearance (Ahmad et al., 2012). The significant reduction in cholesterol levels in the Fen I + LS and Fen II + LS groups, particularly at later stage of experimentation, suggests that LS aids in the restoration of lipid homeostasis, possibly by modulating hepatic enzymes involved in lipid metabolism.

Additionally, the elevated blood glucose levels in fenvalerate-treated groups indicate metabolic disturbances, which could be attributed to stress-induced insulin resistance, altered pancreatic function, or impaired glucose uptake by peripheral tissues. Pyrethroid-induced oxidative stress has been shown to interfere with insulin signaling pathways, leading to hyperglycemia and glucose intolerance (Kale et al., 2016). While LS supplementation resulted in a slight reduction in glucose levels, it did not restore them to baseline, suggesting that while LS confers some metabolic protection, it may not fully counteract pesticide-induced dysregulation of glucose homeostasis.

A significant reduction in total protein levels in fenvalerate-exposed groups further supports the notion of hepatic dysfunction, likely due to impaired protein synthesis and hepatocellular damage. The compromised synthesis of essential proteins such as albumin and globulins could result from fenvalerate-induced oxidative stress and mitochondrial dysfunction, leading to hypoproteinemia (Raina et al., 2008). The partial recovery of protein levels in LS-treated groups indicates that LS may support hepatocyte function by reducing cellular oxidative stress and enhancing protein synthesis machinery.

CONCLUSION AND FUTURE PERSPECTIVES

The findings of this study confirm that fenvalerate exposure induces significant hepatic and metabolic toxicity, as evidenced by alterations in biochemical markers.

The partial hepatoprotective effects of *Lepidium Sativum* suggest its potential role in mitigating oxidative stress, improving lipid metabolism, and supporting hepatic function. However, the incomplete normalization of glucose levels and only partial restoration of protein synthesis indicate that additional mechanisms, such as endocrine and immunological factors, may contribute to pyrethroid toxicity.

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